strated the importance of arterial damage and thrombosis in initiating the tissue damage. Kinmonth and Shepherd[5] supported this conclusion with animal studies, and also showed that surgical sympathectomy and heparinization reduced the area of gangrene of the ear in rabbits. Biopsy evidence indicates that regardless of the various causative factors, thrombosis eventually develops in all cases in which tissue damage occurs. Although no reported study has dealt with intra-arterial injection of promethazine, the development of necrosis and gangrene in our patient probably involved thrombosis of the arterioles. Phenergan is an aqueous solution of 10 per cent promethazine HCl, *p*H 5.3, recommended for use im or iv.[6]

At present, there is no documented explanation for the development of gangrene after intra-arterial injection of promethazine HCl. The effect of any treatment is difficult to evaluate, since the condition is not clearly understood. However, in treating our patient we could not use heparin. Stellate ganglion block improved the condition temporarily, but could not prevent the development of gangrene.

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Pulmonary Embolism Following Spinal Anesthesia: Report of a Case

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Spinal anesthesia is frequently accompanied by decreases in blood pressure of various degrees. Decreased peripheral resistance, pooling of blood, and diminished cardiac output have been shown to be responsible for the hypotension.[1] The following case history documents sudden hypotension accompanying spinal anesthesia which was relatively resistant to the usual modes of therapy and was associated with cardiac arrhythmia and pulmonary embolism.

Report of a Case

A 42-year-old Negro man was transferred from another hospital with an intracapsular fracture of the left femoral neck, suffered in a fall five days earlier. Treatment had consisted of bed rest, Buck's traction, and analgesics. The patient had been medically retired from military service because of a seizure disorder, related to chronic alcoholism. Maintenance doses of diphenylhydantoin and phenobarbital had kept him seizure-free for two years. He denied cardiorespiratory disease. No other significant facts were documented by medical history.

Physical examination was within normal limits except for the hip fracture. Chest x-ray disclosed no abnormalities. An electrocardiogram showed nonspecific ST–T wave changes and sinus rhythm. Open reduction of the fracture was scheduled for the following day.

After premedication with morphine sulfate, 10 mg, hyperbaric spinal anesthesia was administered using 12 mg of tetracaine, while the patient remained awake and cooperative. The sensory skin anesthetic level was determined to be T-8, and the left leg was elevated for surgical preparation. Blood pressure was 140/90 mm Hg, and the pulse was regular at 80 beats/min. A total of 4 ml of Innovar was administered in increments for further sedation. Within five minutes the blood pressure was inaudible; then it was determined to be 60 torr by cuff occlusion and palpation of the

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radial pulse, which was weak, rapid, and somewhat irregular.

The patient's legs were elevated and 800 ml of 5 per cent dextrose in Ringer's lactate solution was infused rapidly, while oxygen was administered by mask. No change in blood pressure was observed. Ephedrine sulfate was administered iv in 25-mg increments, and after a total of 75 mg, systolic blood pressure was palpable at 80 torr. A cardioscope tracing revealed tachycardia and an irregularity.

The patient was moved to the recovery room, and an electrocardiogram confirmed atrial fibrillation with a rapid ventricular rate. Central venous pressure was 14 cm H_2O. Blood pressure stabilized at 80/60 torr. A total of 1.0 mg digoxin was given, iv, in two hours, while the patient remained sedated but responsive. Within four hours he was more alert and denied chest pain or dyspnea. There was spontaneous reversion to sinus rhythm, but the tachycardia and ST–T wave changes persisted. Chest x-ray remained normal. The temperature increased to 101.4 F.

Approximately 44 hours after the onset of atrial fibrillation, the patient was awakened by right anterolateral chest pain of a pleuritic type, accompanied by cough and hemoptysis. A repeat electrocardiogram showed right axis shift, cor pulmonale, and deep S waves in leads 1, V_2, V_3, and V_4. Analysis of blood gases showed arterial hypoxemia. Portable chest x-ray was normal, but a radioisotope scan suggested pulmonary embolism. Serum enzyme studies supported the diagnosis. Heparin anticoagulation and supportive care resulted in a gradual decrease in symptoms, electrocardiographic changes regressed, and a repeat pulmonary radioisotope scan 25 days later disclosed no abnormalities. Heparin was discontinued and open fracture reduction was rescheduled. The patient tolerated general endotracheal anesthesia and the operation without further complication.

Discussion

Atrial fibrillation is not usually caused by hypotension alone, and when this arrhythmia appears, other etiologies should be suspected. Beta receptor stimulators can produce atrial fibrillation; however, this patient's pulse was irregular before the administration of ephedrine sulfate. Paroxysmal atrial fibrillation can be produced by pulmonary embolism, particularly in abnormal hearts, and may be the first manifestation of right heart strain.[2, 3]

Minor degrees of pulmonary embolism may produce few circulatory or pulmonary disturbances, and can go unnoticed.[4] Frequently, dyspnea, anxiety, and tachycardia occur, although sedative drugs and narcotics can obtund these symptoms.[5] Only when pulmonary infarction has occurred do pleuritic chest pain and hemoptysis develop. The time interval between embolic occlusion and infarction can vary from hours to days. Increase in the size of the original embolus or recurrent embolization may be responsible for the delay.[6]

Persistent tachycardia following conversion to sinus rhythm and an increase in fever support the conclusion that the patient experienced pulmonary embolism in the operating room, prior to the onset of pleuritic pain and hemoptysis 44 hours later. Whether venodilatation, as a result of spinal anesthesia, contributed to the propagation and central migration of a thromboembolus is a matter of speculation, and rather doubtful.

Any bedridden patient who suddenly develops dyspnea, tachypnea, tachycardia, unexplained fever, or arrhythmia, with or without classical symptoms, must be suspected of having developed pulmonary embolism. Proposed surgery should be postponed. Chest x-ray, electrocardiogram, serum enzymes, and arterial blood gas analysis may help support the diagnosis, but can be nonspecific. Pulmonary radioisotope scan is a simple and widely used procedure for confirming the diagnosis, and should be accomplished early after symptoms and signs suggestive of pulmonary embolism appear.

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